Oleanolic acid

Cat. No.: HY-N0156

CAS No.: 508-02-1

Molecular Formula: $C_{30}H_{48}O_3$

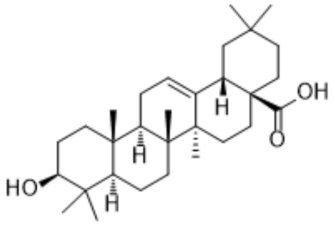
Molecular Weight: 456.7

Target: Autophagy; Endogenous Metabolite; HIV; MAP3K

Pathway: Autophagy; Metabolic Enzyme/Protease; Anti-infection; MAPK/ERK Pathway

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMF : 45.45 mg/mL (99.52 mM; ultrasonic and warming and heat to 60°C)
DMSO : 5 mg/mL (10.95 mM; Need ultrasonic)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.1896 mL	10.9481 mL	21.8962 mL
5 mM	0.4379 mL	2.1896 mL	4.3792 mL
10 mM	0.2190 mL	1.0948 mL	2.1896 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 0.5 mg/mL (1.09 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 0.5 mg/mL (1.09 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.5 mg/mL (1.09 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Oleanolic acid (Caryophyllin) is a natural compound from plants with anti-tumor activities.

IC_{50} & Target

ASK1

In Vitro

Oleanolic acid (OA) suppresses the proliferation of lung cancer cells in both dose- and time-dependent manners, along with an increase in miR-122 abundance. CCNG1 and MEF2D, two putative miR-122 targets, are found to be downregulated by OA

	treatment [1]. OA induces autophagy in normal tissue-derived cells without cytotoxicity. OA-induced autophagy is shown to decrease the proliferation of KRAS-transformed normal cells and to impair their invasion and anchorage-independent growth[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Mouse model experiments also demonstrat that OA suppresses the growth of KRAS-transformed breast epithelial cell MCF10A-derived tumor xenograft by inducing autophagy [2]. Activation of MAPK pathways, including p-38 MAPK, JNK and ERK, is triggered by OA in both a dose and time-dependent fashion in all the tested cancer cells. OA induces p38 MAPK activation promoted mitochondrial translocation of Bax and Bim, and inhibits Bcl-2 function by enhancing their phosphorylation. OA can induce reactive oxygen species (ROS)-dependent ASK1 activation, and this event is indispensable for p38 MAPK-dependent apoptosis in cancer cells[3].It is also proved that p38 MAPK knockdown A549 tumors are resistant to the growth-inhibitory effect of OA[3]. In OA-treated EAM mice the number of Treg cells and the production of IL-10 and IL-35 are markedly increased, while proinflammatory and profibrotic cytokines are significantly reduced[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Acta Pharm Sin B. 2024 Mar 18.
- PLoS Biol. 2024 June 27.
- Pharmacol Res. 2024 May 9:204:107208.
- Food Chem. 2022: 134807.
- Phytomedicine. 2023 Nov 11, 155208.

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REFERENCES

[1]. Zhao X, et al. Oleanolic acid suppresses the proliferation of lung carcinoma cells by miR-122/Cyclin G1/MEF2D axis. Mol Cell Biochem. 2015 Feb;400(1-2):1-7.

[2]. Liu J, et al. Oleanolic acid inhibits proliferation and invasiveness of Kras-transformed cells via autophagy. J Nutr Biochem. 2014 Nov;25(11):1154-60.

[3]. Liu J, et al. p38 MAPK signaling mediates mitochondrial apoptosis in cancer cells induced by oleanolic acid. Asian Pac J Cancer Prev. 2014;15(11):4519-25.

[4]. Martín R, et al. Oleanolic acid modulates the immune-inflammatory response in mice with experimental autoimmune myocarditis and protects from cardiac injury. Therapeutic implications for the human disease. J Mol Cell Cardiol. 2014 Jul;72:250-62.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA